United States
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Animal and
Plant Health
Inspection
Service

Program Aid 1579

Keeping America Free From Foreign Animal Diseases

Lumpy Skin Disease, Sheep Pox, and Goat Pox

Volume
4

Guidelines for Using This Package

This binder contains an integrated suite of educational materials about lumpy skin disease, sheep pox, and goat pox. The package can be used in a formal training setting, where a presenter will show the video tape and narrate the slide show using this black-and-white brochure as the script. Or the materials can be used in a self-study program with the reader progressing at his or her own pace.

Within this brochure, readers will notice that certain paragraphs are preceded by a number. These numbers correlate to the slide set. For example, the lumpy skin disease slides are marked "LS" at the top of each plastic slide frame and numbered sequentially from 1 to 37. The sheep pox and goat pox slides are marked (SP) and numbered from 1 to 42.

If you remove the slides from their protective clear-plastic sleeve (for example, to put them into a carousel for group viewing), please be sure to reposition them in the correct numeric order for the benefit of future users.

This shrink-wrapped suite includes a general video tape, a scientific video tape, and a slide set on lumpy skin disease, sheep pox, and goat pox, plus the brochure you are reading now. If your package is incomplete, please contact the following office for replacement materials:

U.S. Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services, Emergency Programs
4700 River Road, Unit 41
Riverdale, MD 20737–1231

Instructional packages on other diseases are also available and may be requested by writing to the above address. Titles include
Program Aid 1576 African Horse Sickness
Program Aid 1577 African Swine Fever
Program Aid 1578 Contagious Bovine Pleuropneumonia
Program Aid 1580 Malignant Catarrhal Fever
Program Aid 1581 Rinderpest, Peste des Petits Ruminants
Program Aid 1582 Vesicular Diseases

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Lumpy Skin Disease

Definition

1 Lumpy skin disease (LSD) is an acute infectious disease of cattle caused by a poxvirus. The disease is characterized by fever and eruptions of a large number of intradermal nodules of varying size that later undergo an inverted conical necrosis (sitfast).

Etiology

2 LSD is caused by a *Capripoxvirus*. LSD virus (LSDV) is also known as Neethling virus to distinguish it from a herpesvirus that causes pseudo LSD. The Neethling virus is the prototype of LSDV. LSDV is very large—170–260 × 300–450 nm. Other viruses in genus *Capripoxvirus* include the viruses that cause sheep pox and goat pox. LSD, sheep pox, and goat pox are so closely related that they cannot be differentiated serologically. The antigenic relationships and host specificity between these viruses are not clear. Some strains of sheep pox virus will infect goats and protect cattle against an infection by LSDV.

There is one antigenic type of LSDV.

LSDV is stable between pH 6.6 and 8.6.

There is no loss of titer at 37 °C for 5 days.

LSDV is viable at 4 °C for 6 months.

LSDV can be recovered from dried hides after 18 days.

LSDV can be recovered from salted hides.

Effective Disinfectants

3 Common disinfectants are effective. Good choices are the iodophore and chlorine dioxide disinfectants.

History

4 LSD was first recognized in Northern Rhodesia (Zambia) in 1929. Since that time, LSD has slowly spread throughout much of Africa. In the last few years, it has occurred in Egypt and Israel.

Allerton virus infection of cattle in Africa—bovine herpes mammillitis virus elsewhere—*Bovid Herpesvirus*-2—has been misdiagnosed as LSD and caused confusion in vaccine experiments.

Host Range

5 LSD occurs in cattle. There is no report of LSD occurring in wild game animals. It has been suspected that the Cape buffalo might be a reservoir.

Geographic Distribution

6 LSD occurs in Africa. The disease occurred in and was eradicated from Israel.

Transmission and Epidemiology

7 LSD is not a very contagious disease. LSDV has been found in blood for 4 days, in saliva for 11 days, and in semen for 22 days after the onset of fever. The virus is present in skin lesions for 5 or more weeks after viremia. Transmission by close contact has occurred only when animals shared the same drinking bowl.

LSD is spread primarily via biting flies and mosquitoes *(Stomoxys calcitrans, Culex mirificus,* and *Aedes natronius* have been associated with transmission) and by movement of infected animals. The disease spreads along river basins. Outbreaks occur during the rainy season and disappear at the onset of the dry season.

Incubation Period

8 Following natural exposure, the incubation period is 2–4 weeks. After experimental inoculation, the incubation period is 5–12 days.

Pathogenesis

9 At the site of infection, LSDV replication occurs in the epidermis and dermis. The virus then spreads via infected macrophages to local lymph nodes, where it continues to replicate and causes extensive lymphoid proliferation and lymph-node enlargement. Then LSDV-infected macrophages spread throughout the body via the blood. The viremia lasts about 4 days, and there is one "crop" of disseminated lesions. In the body, LSDV can replicate in a wide variety of cells—epidermis, dermis, endothelial, muscle, joint, testis, and serous membranes. The conical necrotic area in the skin—sitfast—may be due to a vasculitis and thrombosis.

Clinical Signs

10 Cattle of all ages can be affected by LSD, but young calves are usually more severely affected. In infected animals, signs range from virtually subclinical infection to severe clinical disease.

Fever can vary from 104 to 107 °F (40 to 41.7 °C).

Within 48 hours of the onset of fever, lumps can appear in the skin, and there will be enlargement of the prescapular, prefemoral, and popliteal lymph nodes.

The lumps in the skin can vary in number from a few to many hundred and in size from a few millimeters to several centimeters and coalesce to form plaques.

The lesions can occur all over the body but tend to occur more frequently in the skin on the neck, the perineum, and the posterior aspects of the thighs. Other sites for lesion development are the oral cavity, conjunctiva, muzzle, turbinate mucosa, glans penis, and prepuce.

Early in the development of the skin lesion, the epidermis appears normal, but the area affected is accentuated because the hair stands more erect.

As the skin lesions develop, the characteristic feature of LSD is that the whole thickness of the skin (epidermis and dermis) is involved. You can determine this by palpation. Also, the lesions are circular and well demarcated.

In some animals, the lesions will not progress and will eventually, in months, resorb. In other animals, the centers of the lesions will become necrotic, be walled off, but remain in place. The lesions form a sequestrum within the nodule called a sitfast, which is characteristic of LSD. If the sitfast is removed, a deep ulcer forms.

The number and size of the lesions in the skin reflect the severity of the illness. However, in some very severe cases seen following experimental intravenous inoculation, animals have been observed to die with only internal lesions.

In severe cases, the appearance of skin nodules is accompanied by salivation, ocular and nasal discharge, and reluctance to move. Severely affected animals tend to lose a lot of weight.

Clinical Signs

11 Cattle in Egypt with natural infections of LSD. Note that the animals are very thin. Experimental animals inoculated with this isolate also lost a lot of weight.

12 Natural infection showing lumps on the side of the neck.

13 Natural infection showing numerous lumps in the skin. Some of the lumps have a dark center.

14 Calf with a severe natural infection.

15 Many lumps ranging in size from small to large. If these lumps are palpated, they will feel thick because of involvement of the skin and dermis.

16 An early stage in the development of LSD lesions on the face. These lesions are red and slightly raised and thickened.

17 A typical well-developed LSD lesion. This is a deep lesion. Note the line of separation around the necrotic center, where a sitfast is beginning to develop.

18 A more advanced stage in the development of sitfasts. The necrotic center of the lesions is black, and there is a well-developed line of separation. Note that even small lesions have a black center.

19 A sitfast has been pulled off. Note the depth of the lesion.

20 Sitfast on a teat. In a milking animal, this might result in a fistula and/or mastitis.

Gross Lesions

21 At necropsy, in addition to the locations of lesions given under Clinical Signs, lesions may be found in the mucosa of the respiratory sinuses, turbinates, trachea, lungs, mouth, forestomachs, serosal surfaces, and joints (synovitis and tendosynovitis).

The typical skin lesion involves the epidermis and dermis and extends into the underlying muscle. When the skin is reflected, the lesion can be seen on the undersurface.

Lesions in the respiratory epithelium are circular, blanched looking, and circumscribed by hyperemia. Lesions in lungs are not as frequent or as obvious in LSD as in sheep pox. In cattle, lesions due to LSDV appear as an area of edema.

22 A section through a lesion over the hock. Note the depth of the lesion.

23 The skin has been reflected. The pink areas on the underside of the skin and in fascia over the muscle are extensions of the pox lesions into the dermis and subcutaneous fascia.

24 The underside of the skin. A necrotic core of the sitfast, which extends through the skin into the subcutaneous tissue, has been sectioned parallel to the skin.

25 Numerous LSD lesions in the nostrils and on the anterior parts of the tubinates.

26 Two well-developed pox lesions on the turbinates. The pale center is surrounded by a hyperemic ring.

27 Well-developed pox lesion in the trachea. The pale center is surrounded by a hyperemic ring.

28 LSD lesions in the lung form a diffuse area of consolidation and edematous interlobular septa.

Microscopic Lesions

The histologic appearance of the skin lesion depends on the stage of LSD infection. In nodules collected soon after eruption, the epithelial cells in the stratum spinosum may be undergoing hydropic degeneration and be coalescing to form microvesicles. There may be eosinophilic inclusion bodies in the cytoplasm.

Under the epithelium, the histiocytic cells are proliferating and form thick cuffs around blood vessels, and there may be thrombosis. Inclusion bodies may be present in the macrophages. There may be scattered necrotic cells in the area.

A similar histiocytic proliferation can occur under lesions in the respiratory epithelium.

Photomicrographs of LSD Lesions

29 Microvesicles in an early LSD lesion.

30 A section of dermis under a large LSD lesion in the skin. Note clusters of inflammatory cells and their relationship to vessels.

31 A higher magnification of a hyperplastic focus. The wall of vessel in the center of the photograph has been infiltrated.

32 The wall of the arteriole appears to be degenerating, and there is a thrombus in the lumen.

Morbidity or Mortality

33 Morbidity associated with LSD varies greatly among herds, ranging from 3 percent to 85 percent. Morbidity most likely depends on the prevalence of the vector and the availability of the cattle to the vector. Mortality is usually low (from 1 percent to 3 percent) and caused by secondary infections.

The economic loss results from secondary infection, which causes debility and weight loss, up to a 50-percent drop in milk production, infertility in bulls, abortion (infrequent), and scarred hides.

Diagnosis

Field Diagnosis

34 LSD should be suspected when a febrile disease in cattle is accompanied by generalized, circular, well-demarcated skin lesions that involve the epidermis and dermis and undergo necrosis.

35 Specimens for the Laboratory

In live animals:

- Early (no necrosis) skin lesions should be biopsied.
- Enlarged lymph nodes should be biopsied with a large needle and aspirated.

At necropsy, collect the following:

- Skin lesions
- Lymph nodes
- Lung lesions

Acute and convalescent serums should be collected 2 to 3 weeks apart.

The biopsy specimens should be shipped on wet ice; if the time to the laboratory will be prolonged, the specimens should be shipped on dry ice, ideally in screw-capped tubes with the lids taped shut.

Biopsies of early and late lesions should be preserved in 10-percent formalin and sent to the laboratory unfrozen.

36

Laboratory Diagnosis

Virus Isolation—Cell cultures of lamb testicle and/or fetal bovine lung tissue are usually used for virus isolation.

LSDV can also be detected in lesions and scabs by direct electron microscopy.

Serology—Antibodies can be detected by virus neutralization or by the indirect fluorescent antibody test.

Vaccination

37

Cattle can be protected by vaccination with an attenuated LSDV vaccine or sheep and goat pox vaccine.

Control or Eradication

38

In endemic areas, vaccinate cattle. In LSD-free areas, do not import cattle from an infected area without proper quarantine.

If LSD occurs in an area usually free of it, place a quarantine around the infected area, slaughter infected and exposed cattle, and clean and disinfect the premises. Consider ring vaccination.

Israel has been the only country to eliminate an incursion of LSD. The eradication was accomplished by quarantine, slaughter of the infected herd, and ring vaccination.

Sheep Pox (SP) and Goat Pox (GP)

Definition

1 SP and GP are acute infectious diseases caused by poxviruses. These diseases are characterized by fever and eruptions of a large number of intradermal nodules of varying size that later may undergo necrosis.

Etiology

2 The etiology resembles that of LSD. The viruses in dry scabs are stable for a long period.

3 Photo of a poxvirus.

Effective Disinfectants

4 Common disinfectants are effective. Good choices are the iodophore and chlorine dioxide disinfectants.

History

5 SP and GP were recognized in the 2d century A.D. They were first recognized as contagious diseases in 1673. SP was studied in depth in 1903 by Borrel. Only in recent years has SP been eradicated from Eastern Europe.

There was an outbreak of SP in Italy in 1983.

Host Range

6 These diseases affect sheep and goats. Inoculated cattle develop subclinical infection.

Geographic Distribution

7 These diseases are present in Africa, the Middle East, the Indian subcontinent, and Southwest Asia.

Transmission and Epidemiology

8 Animals are infected by inhalation of aerosols produced by acutely infected animals and/or dust contaminated by pox scabs. (Dried scabs fall off, and animals running over the area grind up the scabs and create dust.) Animals may also be directly or indirectly infected via abrasions (e.g., abrasions caused by shearing equipment). In experiments, animals can be infected by intranasal, scarification, subcutaneous, or intravenous inoculation. Flies feeding on lesions are potential mechanical vectors.

In areas where SP and GP are endemic, about half of newborn lambs and kids are susceptible to them. The long survival of the viruses in scabs and the continual presence of susceptible animals ensures persistence of the diseases. Herding systems and gathering animals at wells, markets, etc., provide ideal conditions for spread.

Incubation Period

9 The incubation period after natural exposure is usually 4 to 8 days but can be up to 3 weeks.

Pathogenesis

10 The pathogenesis of SP and GP is similar to that of LSD.

Clinical Signs

11 Clinical signs of SP and GP can vary from subclinical to severe. A classic infection develops as follows:

- Initial signs are fever, rhinitis, and increased lacrimation.

- About 2 days later, there are small, red papules in the skin over the whole body. The papules are most easily seen in sparsely wooled or haired areas, such as the axilla, groin, scrotum, vulva, underside of the tail, udder, ears, eyelids, lips, and nostrils. The papules then progress and appear as red, firm, elevated (2 to 3 mm), flattened lesions (plaques). If palpated, such a lesion will feel thick; the lesion is in both the epidermis

and dermis. Next, the epidermal cells in the center of the lesion then undergo hydropic degeneration to form microvesicles. This area then becomes depressed and grey (necrotic). Superficial lymph nodes are greatly enlarged. As the necrotic area dries, it becomes a black scab. The lesion heals slowly, and when the scab comes off, there is a scar.

During the acute stage of the disease, the animal is depressed and anorectic, stands with an arched back, and may have rapid, labored breathing because of lung lesions. The eyelids, lips, and nares may be swollen because of numerous lesions.

Photographs of Clinical Signs of SP

12 Three sheep with acute SP infection. The animals are inactive and depressed. The side of one sheep has been sheared so the extent of skin involvement can be seen.

13 Closeup of a sheared area. All the red areas are developing pox lesions.

14 The first skin lesions of pox are small, red papules.

15 The lesions then enlarge to form plaques. In this photo, the centers of the plaques are red because of vesiculation and beginning necrosis. When the lesions at this stage are palpated, they will feel thick because the whole thickness of the skin and dermis is affected.

16 The centers of the lesions appear grey because of necrosis in the upper layers of skin.

17 The lesions in this photograph are old. The necrotic centers have dried to form black scabs, and swelling around the lesions is decreasing.

The Lesions in GP

18 The centers of the skin plagues are depressed and more dark than the surrounding area because of vesiculation and necrosis.

19 The side of this goat has been clipped to allow observation of the severity of skin involvement.

20 Closeup of the skin lesions. The whole surface of the plaque is grey as a consequence of necrosis. The plaques have a hyperemic border.

21 The plaques under the tail have vesiculated and necrotic centers that have eroded.

22 A group of GP plaques. These lesions will feel thick because the whole thickness of the skin and dermis is affected.

23 A cut surface of a skin to show the depth of the pox lesion.

Gross Lesions

24 Skin lesions are described in the preceding slides. When the skin is reflected, the lesions in skin can be seen as red areas extending into the subcutaneous tissue and even the cutaneous muscle. Lymph nodes draining affected areas of skin will be greatly enlarged.

Pox lesions may also occur in the mucosa of the respiratory sinuses, turbinates, trachea, lungs, mouth, forestomachs, prepuce, and testicles.

Pox lesions in respiratory epithelium are circular, blanched looking, and circumscribed by hyperemia.

The appearance of pox lesions in the lungs will depend on the interval between infection and death. Regardless of the interval, the lesions will be focal and uniformly distributed throughout the lungs because they are of hematogenous origin. In severe cases, the lungs will be distended and firm. Early lesions will be focal, red, and edematous and be so numerous as to almost coalesce. As the lesions develop, they become more nodular and white, and the lung tissue between the nodules appears more normal.

Pox lesions in the forestomachs will form raised areas in the epithelium.

Photographs of Gross Lesions of SP in the Lungs

25 The red areas are the first stage of pox lesions in the lungs. Note the fairly uniform distribution of lesions; this is due to a hematogenous origin of the infection. A lung like this one will be quite distended and feel firm.

26 This lung has fewer lesions, and their edges are more defined. These lesions are older and more developed.

27 Closeup of well-developed lung lesions.

28 When the lung lesions mature, they become firm, white nodules. Note the uniform distribution. These nodules can be confused with granulomas, but granulomas are usually less numerous. Granulomas from lung worms are often distributed in the dorsal part of the lungs.

Microscopic Lesions

To study the development of pox lesions histologically, collect biopsies from the time when the lesion appears as a red, firm plaque to when the center of the lesion becomes depressed and grey. It is during this period that there may be acanthosis, hydropic degeneration, microvesiculation, and eosinophilic intracytoplasmic inclusion bodies in the epithelium. The other distinctive feature of a pox lesion is in the dermis and subcutaneous area under the epithelial lesion. Here there will be a proliferation—particularly around blood vessels—of large, histiocytic-type cells that have nuclei with marginated chromatin, hydropic degeneration, and intracytoplasmic inclusion bodies. These cells are called celles claveleues, Borrel cells, or pox cells.

Photomicrographs

29 A GP lesion in the skin at the plaque stage before necrosis. The epithelium in the center of the plaque has numerous microvesicles.

30 In a higher magnification of the microvesicles, a few eosinophilic intracytoplasmic inclusions can be seen.

31 A section of dermis under a well-developed GP lesion. Note the very prominent thickening of vessels.

32 A higher magnification of a vessel in slide 31. The vessel wall is thickened by hyperplasia of a large histiocytic-type cell. This type of cell is characteristic of a pox infection.

33 A section of dermis under a well-developed GP lesion. There is an inflammatory cell infiltrate around the vessel and in the vessel wall and possibly a mass of cells in the lumen.

34 An early stage in the development of an SP lesion in the rumen. There are microvesicles in the epithelium.

35 An early SP lesion in the lung. The lung is consolidated because of enlargement of alveolar epithelial cells and a mild inflammatory cell infiltrate.

36 These SP lesions in the lung appear older because there are accumulations of necrotic cells in the alveoli and bronchioles.

Morbidity and Mortality

37 Morbidity from SP or GP can be 80 to 100 percent.

The severity of infection varies with the strain of the virus and the breed and age of the animals. In adult animals, the disease can range from a subclinical infection to an infection with high (50 percent) mortality. Generally, adult animals have a milder infection than young animals. In lambs and kids under 1 month old, mortality rates can approach 100 percent.

Diagnosis

Field Diagnosis

38 SP and GP should be suspected when animals have fever and generalized intradermal skin lesions that undergo necrosis.

Specimens for the Laboratory

Early skin lesions as described above should be collected by biopsy for virus isolation (lesions should be refrigerated) and histologic examination (lesions should be fixed in formalin).

Enlarged lymph nodes can be needle-biopsied and aspirated for specimens for virus isolation.

Dry scabs should be collected for virus isolation and direct electron microscopy.

Acute and convalescent serum samples should be collected 2 to 3 weeks apart.

At necropsy, skin lesions, enlarged lymph nodes draining the skin, and lesions in the upper respiratory tract and lungs should be collected for virus isolation. A complete set of tissues should be collected and placed in 10-percent buffered formalin.

Specimens for virus isolation should be shipped on wet ice. If transit time to the laboratory will prolonged, the specimens should be shipped on dry ice, ideally using screw-capped tubes with the lids taped shut. Do not freeze formalin-fixed specimens.

Laboratory Diagnosis

40 **Virus Isolation**—Virus isolation is done using cell cultures of fetal or neonatal lamb testicle and kidney tissue. SP and GP viruses can also be detected in lesions and scabs by direct electron microscopy.

Serology—Antibodies can be detected by a virus neutralization test or by the indirect fluorescent antibody test.

41 **Vaccination**—The two most used attenuated vaccines are the Romanian strain and the Kenya O 180 strain. Inactivated vaccines do not provide solid, long-lasting immunity.

Control or Eradication

42. In areas where SP or GP is endemic, the animals should be vaccinated.

To prevent the introduction of SP or GP into a disease-free area, prohibit the importation of sheep, goats, and sheep and goat products (meat, wool, hair, hides) from an infected area unless there is proper testing, quarantine, or decontamination.

If SP or GP occurs in a disease-free area, place a quarantine around the infected area, slaughter the infected and exposed animals, and clean and disinfect the premises. Also, consider ring vaccination.